surface expression, we examined DeltaD distribution after Notch signaling was blocked by a gamma-secretase inhibitor, DAPT or by morpholinos directed against Notch. Both manipulations resulted in exaggerated expression of deltaD. In DAPT-treated embryos some DeltaD accumulated on the plasma membrane, however, most was in cytoplasmic puncta. In contrast, there was a significant increase in DeltaD surface expression in Notch morphants. This suggests that exaggerated expression of deltaD transcripts resulting from failure of Notch signaling per se contributes minimally to increased surface expression. Furthermore, the significant increase in Delta surface expression in Notch morphants suggests that endocytosis of DeltaD is not only dependent on Mib function but also on interactions with Notch.

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Program/Abstract # 118
Targeting of Sanpodo to asymmetric pericentrosomal early endosomes regulates Notch signaling in Drosophila sensory organ precursor cells
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Asymmetric cell division is an evolutionarily conserved strategy to generate cell fate diversity during development. In *Drosophila*, dividing sensory organ precursor (SOP) cells segregate the membrane-associated cell fate determinant Numb, an antagonist of the Notch pathway, asymmetrically into the pIIb daughter cell. Notch signaling in the pIIb sister cell, pIIa, requires Sanpodo, a transmembrane protein. We showed previously that Numb is required for targeting Sanpodo to vesicles in the pIIb cell cytoplasm. Using in vivo imaging of a Sanpodo-GFP fusion protein, we now show that Numb is required for Sanpodo to be efficiently sequestered from the plasma membrane to a subpopulation of pericentrosomal Rab5-positive early endosomes in the pIIb cell within 10 min of the completion of SOP asymmetric cell division. Blocking formation of the early endosome compartment by inhibiting endocytic vesicle fusion causes failure of Sanpodo accumulation in large endosomes and results in cell fate switching of the pIIb cell due to increased Notch activity. We are currently exploring the mechanism of asymmetric recruitment of Rab5-positive early endosomes to the centrosome in the pIIb cell and are determining the temporal requirement for Notch signaling following SOP division by using a temperature-sensitive allele of Notch. We hypothesize that Notch signaling is regulated by sorting of Sanpodo, and perhaps other Notch pathway components, from the plasma membrane into an asymmetric endocytic node that forms around the centrosome in the pIIb daughter cell shortly after SOP cell mitosis.

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Program/Abstract # 119
Characterization of the protein localization of pyramus and thisbe, *Drosophila* FGF ligands
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Fibroblast Growth Factors (FGFs) are involved in important developmental processes including mesoderm induction and patterning, organ formation, and neuronal differentiation. A pressing question in the FGF field is how specificity is achieved and distinct cellular responses accomplished with so few receptors compared to ligands. The FGF family of ligands has two relatively new members in *Drosophila*, *pyramus* (*pyr*) and *thisbe* (*ths*), both of which activate the *heartless* (*htl*) FGFR. Two FGF ligands, *pyr* and *ths*, activate the *htl* receptor, a simplified system compared to the situation in vertebrates where often 4 or more ligands can activate a single receptor. We use this model to investigate how signaling by *pyr* versus *ths* could give rise to different cellular responses. We made HA-tagged versions of Pyr and Ths proteins and have shown these proteins to be functional in the embryo. These epitope tags have allowed us to visualize the proteins using GAL4 lines to overexpress them in the mesoderm (*twist*) and neuroectoderm (*69B*). These patterns will be compared with those in a *htl* deficiency background to see if the ligand–receptor complex is required to stabilize the protein. Further investigation of the proteins in a *pyr/ths* deficiency background will allow us to see whether the protein localization of one ligand is affected when the other is not present.

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Program/Abstract # 120
Developmental regulation of the cell cycle transition from genomic to site-specific DNA replication in *Drosophila*
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Cell-cycle regulation plays a crucial role in the development of virtually all multicellular organisms. *Drosophila* oogenesis is an excellent model system to study developmental regulation of cell cycle programs, because the epithelial follicle cells of the egg chamber undergo two temporally regulated cell-cycle transitions. The first switch, from the archetypical mitotic cycle to the endocycle, a cell-cycle variant in which DNA is replicated but cell division is bypassed, is induced by Notch activation. The regulation mechanisms of the second transition, from the endocycle to a site-specific gene amplification cycle, are unclear. Here we show that Notch activity is down-regulated at stage 10B, when the follicle cells transit from the endocycle to site-specific gene amplification. Using a constitutively active form of Notch, Notch Intracellular Domain (NICD), we show that extended Notch signaling in follicle cells causes defects in the transition to the site-specific gene amplification cycle.